Claims.

I claim:

Claim 1. The general process of the AIDS development by the HIV lentivirus, characterized by the following characteristics:

(1) There are the two obligatory phases of the AIDS development, coinciding with the 2 type PRINCIPAL obligatory contaminations.

(2) Only the mobile macrophages are contaminated during their movement during the 1st contamination.

(3) This movement is initiated by the cytokine secretions, which provoke: (a) the corresponding adhesion molecule appearance in the organs-targets; (b) the β-chemokine secretion serves as the chemoattractive substances for the macrophage movement, directed to the organs (encephalite origin in the case of the intensive brain contamination) wherein, justly, these β-chemokines with their corresponding receptors coinfect the macrophages/monocytes by HIV.

(4) The relativily weak HIV concentrations can make the 1st contamination due to the patching, capping and endocytosis of many receptors, participating in the cell mobility, provoking the aggregate creations ("critic mass"), including the syndecan molecules, which make the microfilament attachment to the aggregated receptors, conducting (at the end) to the endocytosis and HIV contamination.

(5) The anti-envelope (HIV) antibodies appear later in avoiding the interactions with the viral particles of the original contamination destructed already (the basis of the 2nd contamination necessity).

(6) The 1st nonproductive HIV entry takes place with the viral replicated uninegrated DNA with the heterogeneities of DNA and the corresponding proteins, with the intracysternal A particle (IAP) presence and with the heterogenous pattern of the antibodies against the corresponding viral proteins.

(7) Such IAPs make the very weak viral pseudocontaminations of other cells due to the RNA and reverse transcriptase that they contain.

(8) The heterogeneity of the env viral proteins is generally absolutely necessary for the HIV consecutive productive contamination due to an increased complex number of the Fc fragments of the heterogenous (also) aggregated anti-env antibodies with the cellular Fc receptors that facilitates the virus penetration into the cell at the 2nd phase.

(9) The viral env protein heterogeneity often takes place at the carbohydrate attachment sites (O- and N- chains) and consequently the carbohydrate pattern of the env proteins must be especially heterogenous.

(10) The mortality acceleration at the AIDS phase takes place due to the contamination facilitation at this phase by the complement receptor (C1qR), interacting with the C1q factor, activated by the anti-opportunist agent antibodies.

(11) The weak correspondence between the carbohydrate patterns of the Fc-

receptor cell machinery (of chimpanzee) and the HIV prevent (logically) the 2nd contamination and the AIDS phase (chimpanzee "immunity").

(12) The baby macrophages are more active than those of adult and the baby immune system can already product the antibodies very quickly after the birth, but the 2nd AIDS phase can take place only since ~3 months of age due to the created carbohydrate pattern correspondence between the baby macrophage Fc receptor machinery and HIV virus and, generally, the 2nd contamination can take place due to the very weak transmitted dozes.

(13) The 1st macrophage contamination is made by the macrophage- tropic clones as well the 2nd contamination with also T-cell contaminations, and the T cell- tropic clones provoke the creation of syncytia that undergo the regulated and accelerated apoptosis and the phagocytosis (by macrophages) by the relatively small, almost unvisible, quantities.

(14) Principally, the vaccination must aggravate the contamination due to the antibodies, that help to the contamination, but due to the vaccination with the homogenous envelope proteins, the strong productive contamination is more problematic and these homogenous antibodies can make some decrease of the viral particle quantity (precipitation) and also some blocking of the 1st entry although in the case of the powerful 2nd HIV entry (with CD4 receptor help) there are the dangerous spontaneous re-enterings.

(15) The restricted HIV-2 contaminations take place because of a weaker variability of the viral proteins during the 1st contamination and a stronger differences between the host and virus carbohydrate patterns.

(16) The discrete switching signal due to the specific interactions between the CD4 molecules and the viral envelope proteins, important for AIDS development, are determined by a more general biological molecular processes.

Claim 2. The principal characteristics of the AIDS development process /(a): there is the 1st productive contamination with the cell motility utilisation; (b) the infection increase depends on the antibodies; (c) there is the protein heterogeneity during the 1st contamination, necessary for the heterogenous antibody production, obligatory for 2nd contamination/ according Claim 1 characterized in that a number of other viral Families possesses them.

Claim 3. The viral exterior proteins (envelope or capsule) /Art.52(4) EPO, Art.2(2) AT (law of 1970)/ /for manufacture of medicaments pour vaccinations against viruses (like influenza A-influenza/pneumonia)/ with one (anyway with minimal possible quantity) viral neutralizing epitope and/or one (minimally possible) viral homogenous clone that must be taken for the immunization characterized in that the increase of the virus contaminations is minimal (zero) because the corresponding anti-viral antibodies could increase the cell contaminations with the Fc receptor help wherein the heterogeneity of these antibodies is, generally, obligatory for the virus entry according Claims

1(8,10,14) and 2.

Claim 4. The substances /Art.52(4) EPO, Art.2(2) AT (law of 1970)/ (for manufacture of medicaments against encephalites), that stop the macrophage motility (as antibodies against the β-chemokine receptors), characterized in that this motility is obligatory for the contaminated macrophage entering into brain to cause the encephalites (as those of CMV origin) according Claims 1(2,3) and 2.

Claim 5. The kit of preparations for the determination of the real titers of the different viruses, with the action, similar to the two HIV phases, according Claims 1(1-5,8 /and Art.52(4) EPO and Art.2(2) AT- law of 1970/ characterized in that for the 1st phase determination: one creates the concentration gradients of the chemokines (as β-chemokines) to switch the macrophage mobility to approach to the conditions of the 1st contamination in vivo, and for the 2nd phase contamination: one utilizes the heterogenous anti-env antibodies (similar to those in vivo) to approach to the conditions of the 2nd contamination in vivo.

Claim 6. The real characteristics of the signalling events that follow the process of the signal switching, produced by the interaction between gp120 and CD4 molecules of the "general process of AIDS development by HIV lentiviruses" of claim 1, are established from the general fundamental processes of the protein foldings and recognitions in the cells, characterized by following caracteristics:

(1) There are the chaperon specializations for each glycosylation type: N-, O- and GAG-, that determine the Universal specialities of a limited chaperon number for a protein enormous number by their carbohydrate chains, specialized due to the law of the homologous intercarbohydrate interactions.

(2) There are the two principal pathways of the protein foldings: endoplasmic reticulum→Golgi and in cytoplasm.

(3) The calnexin (chaperon of ER) is monoglycosylated and it is attached to the N-monoglycosylated proteins after the elimination of two other glucoses.

(4) The calnexin makes the complexes with the calreticulin (ER) due to their homologous O-carbohydrates of their similar structures wherein it is the calreticulin that is responsable for the complex with the BiP and grp 94 chaperons later.

(5) During the 1st "trip" in Golgi (ER→Golgi→ER→Golgi), (a) the proteolysis of the terminal N-end (or C- one) (creation of the peptide, named , Du-2T- like) must take place due to the convertases in Golgi; (b) the gag glycosylation must (can) take place.

(6) The creation of the definitive complex, moreover in ER, of the gp96/grp94 chaperon ("boat") with the BiP, calreticulin, p50-like proteins (GAG specificity), peptidyl-prolyl isomerase (PPI) and protein-disulfide isomerase (PDI) (principally) must take place.

(7) The cytoplasmic folding of the polypeptides, yet attached to the ribosomes in cytoplasm, takes place.

(8) The Hsp70 chaperons (specialized for O-chains) and TRiC chaperons (specialized for Core 2 O-chains, the chaperon of beginning) attach to the elongating peptide near (on) the ribosome.

(9) The hsp90 chaperon serves as "the boat" (in cytoplasm) for other chaperons, including the p50 chaperon, specialized for the GAG- chains (equal to cdc 37), hsp70, PPI (but without PDI), phosphatase (pp5), the CK-II and (in special case) the steroid hormone receptors).

(10) The hsp90 complex proteins (FKBP type) with the protein phosphatase and CK-II (proteins of complete "boat") (serving to the synthesis of the signal proteins of the "PKC" vesicle transporting machinery, having the GR methylated peptide at the C- or N- ends of preproprotein) all go to the nucleus (with these signal proteins) to attach to the preribosomes in nucleus (nucleole) to leave it at the end of G1 (or between signals of "GO").

(11) The hsp90 complex proteins ("boat" type CyP) do not go to the nucleus and serve to the cytoplasm folding of the "purely" cytoplasmic proteins without the GR peptides and without the "trip" into the nucleus.

(12) The Erp61 (grp58) protein is not the chaperon (of PDI family), but it is clearly the vesicular PI-PLC-α, that is well active on "PKC" vesicles, although only after the limited proteolysis, and it is responsable for the phosphatidylinositol-4,5-biphosphate (PIP_2) molecule hydrolysis on the "PKC" transporting vesicles during their spontaneous stopping (like with oxygene cutting) during their functioning at G1 cycle phase or "GO", leading to, at the end, irreversibility of their definite stopping (famous apoptosis!) due to the stock exhaustion of these PIP_2, absolutely necessary for the "PKC" transport vesicle movement (IA,§6; Parts VII,XI), where such irreversibility (apoptosis) could be also due to the stock exhaustion (of proteins, attached with GR peptides on cytoplasmic ribosomes) of the proteins of the "PKC" transporting vesicle machinery.

(13) Particularly, the cytoplasmic steroid receptors "wait" the hormone in the hsp90 "boat" complex (type FKBP) wherein the steroid signal provokes the limited proteolysis (by CL) of the proreceptor and its liberation (with pH increase and protein phosphatase activation).

(14) Generally, the active state with the tension, necessary for the energy stocking during the protein functionings, is made with the two PPI enzymes, that make the two coupled transitions (prolyl trans→cys of two prolyl neighbouring residues with the weak difference of the energies between these positions) wherein the return is impossible because the one sole transition is uneffective for the return and two transitions (at the same time!) are improbable.

(15) In the case of the ER-Golgi pathway, there is the protein foldings with the help of the proteolysed propeptide (from N- or C- ends), attached to the macromolecules and hidden by the two homologous interacting carbohydrate chains, wherein the folding

is accomplished with the distortion mechanism by the tension with the PPI and PDI help wherein with the artificial peptide loss in vivo ("scarpie" form) there is the pathology.

(16) In the case of the cytoplasmic protein foldings, the molecules of the "PKC" transporting vesicle machinery, having the GR peptides and making the "trip" into nucleus there is, firstly, the new chaperon assembling (based on the "boat" hsp90 /type FKBP/) with the preproteins, having the GR peptide, in the nucleus (nucleole) in constructing the preproribosomes, that after the activation (at G1 end) go into cytoplasm, wherein there is (with signal /G1 beginning/) the folding of the new proteins of the machinery with the PPI (FK-506 sensible) help, where the GR prepeptide plays a role, during folding, for the attachment to the ribosomes and later, in the "ready" form, for the "adress" of the destination with the liberation of the 2 interacting (firstly intramolecular interaction) carbohydrate chains, for ,already "definitive", intermolecular interactions, with the 2 (also "intra"intreracting before) glycochains of the target, correspondingly.

(17) In the case of the "pure" cytoplasmic proteins (representing many enzymes) (without "trip" into nucleus), the folding is necessary for the activity (as enzymatic for the tension energy stocking) wherein the propeptide (N- or C-) is necessary for the folding (tension energy) with the PPI (CyP-like) help and where the peptide is not important for the enzyme activity although its association must "abate" definitively the homologous carbohydrate chain dissociation to interact specifically with the chains of the target (adress) of the cytoplasmic proteins.

(18) The proteins (including enzymes) that can be folded with such chaperons must obligatory have their primary structure with the two nearby prolines, corresponding "Du-2T" proteolysable peptides in the proprotein (including the C-end possibility) and corresponding amino acid cluster, permitting the posttranslational modifications with the couple (at least) of the carbohydrate chains, possessing their intramolecular homologous interactions, hiding moreover the "Du-2T" peptide.

(19) The proteins, submitted to this general fundamental process of the protein foldings possess the characteristics for the general recognitions ("adresses"): the couple of their carbohydrate chains being in the intramolecular intercarbohydrate homologous liaison can recognize justly the homologous couple of the (also in intramolecular interactions) carbohydrate homologous chains and provoke the signal beginning with the 2 inter molecular (already) intercarbohydrate hydrogen homologous liaisons, that is "abated" definitively by the corresponding "Du-2T" peptide dissociations.

(20) Particularly, the real characteristics of the signalling events in the process of the AIDS development are established from this (established here) general processes of the protein foldings and recognitions: the couples of the interacting carbohydrate chains of the CD4 molecules (O-chains near the hinge regions) and C2 and V3 regions of gp120 can recognize each other with the switching of the dissociations of these INTRAmolecular bonds for the establishment of the INTERmolecular intercarbohydrate

hydrogen homologous liaisons between CD4 and gp120 chains already, that is "abated" definitively by the dissociation of the corresponding "Du-2T"-like proteins wherein as a result of the strong conformational change, the other intramolecular liaisons are broken, leading to the agglomerate creations with the membrane melting due to the intercarbohydrate dehydration.

Claim 7. The little proteins (peptides) Du-2T like ("Du-2T") of the synthesized, by ER→Golgi, proteins, including all membranous receptors (as CD4 or gp120) of claims 1(16),2 and 6 characterized in that they are obtained from N- or C- proprotein ends after the limited proteolysis, they are hidden at the protein surface by the 2 carbohydrate chains with the homologous intercarbohydrate interactions, they are situated near the site of the 2 prolines and S-S bond(s) (source of the irreversible folding with tension) and its dissociation changes the state of the general structure of these proteins profoundly and discretely in liberating the carbohydrate chains for the intermolecular interactions (for formation of the complex aggregate as in the case of the membranous receptors or/and activation of other molecules) like these real little proteins (peptides) for IgG (and also for Fc receptors and receptors for antigen), characterized in that they are situated in the CH2 region and they dissociate after the hapten (antigen) interaction with the active sites because of the consecutive dissociation of the intramolecular coupled interactions of the covering homologous chains in liberating these immunoglobulin chains from already intermolecular interactions: with N-carbohydrate chains of the complement (C1q) (in activating it) or with the carbohydrates of other receptors: Fc and for antigen and with other carbohydrate chains of plasma membrane (PM); like the little real proteins (peptides) (although exceptionally special) Du-2T- like for the molecules MHC class I, characterized in that they are found in "the active site", created by the α1 and α2 domains of the MHC α-chain with the several prolines of these subdomains wherein "the dissociation" of this peptide (Du-2T -like) in the active site of the TRC provokes the strong change of the general structure of the MHC-I α chain, where the sole N-chain carbohydrate (without pair at invariable site) of this subunit can already interact with the corresponding carbohydrate chain (of the invariable site) of the TCR α-chain (also without pair) to switch the "Du-2T" dissociation from the MHC α3 domain with the 2 covering carbohydrate chains, the dissociation, from TCR, of its "Du-2T" (also there is the presence of the prolines, S-S and two symmetric N-carbohydrate chains) (very exceptional presence of the charged amino acids in the intramembranous domains of all components of TCR facilitates the conformational changes due to instability) and the mutual intermolecular interactions of the O- and N- chains of MHC with those of TCR and the CD8 molecules (having own "Du-2T") and between the carbohydrate chains of the same PM (during the complete signal); and like the functional GPI-anchored proteins (so called priones), synthesized by ER→Golgi (in complex aggregate of the classical complete signal) that make the self-aggregation in pathology (diseases

as Mad Cow or Jacob-Creutzfeldt) (this time, without signal already, but like during the signal!), due to the interactions with the forms of these proteins where the "Du-2T" is dissociated already and the carbohydrate chains (hiding) are free for already homologous intermolecular aggregation, facilitating already the dissociation (facilitated also generally!) the dissociation of the "Du-2T" of other molecules.

Claim 8. The large number of the different little proteins (peptides) (Du-2T-like ="Du-2T") according Claims 1,2,6 and 8 for the manufacture of the medicaments /Art.52(4) EPO, Art.2(2) AT (Law 1970)/ against (1) the undesirable process of the complement activation; (2) the cell lysis; (3) the action of the particular membranous receptors; (4) the action of the parasites as the viruses and also bacteria, protozoans, mashrooms; (5) the creation of the dangerous priones (so called aggregated "scarpie" form) during the diseases as Mad Cow or Creutzfeldt-Jacob; (6) the protein aggregation in solutions (in blood included) characterized in that the simple introduction of these "Du-2T" proteins must stop the corresponding undesirable processes in preventing the dissociation of their native analogues where such dissociation would provoke the harmful signalling including the pathological diseases /(1)-(5)/ or the intermolecular glycoprotein aggregation in solution (6).

Claim 9. The well charged affine molecules (as antibodies) against the distinct HIV surface molecules (or those of other viruses and other parasites like bacteria or mashrooms , or against distinct active sites of the surface antibodies, receptors for antigens (B-cells) and/or TCR producing the harmful anti-HIV-env antibodies (or similar harmful antibodies against the other viruses), the harmful auto-antibodies (as anti-HIV-gag or those in rheumatic diseases) or the harmful allergic antibodies, characterized in that these strong, localized precisely, specifically charges perturb the intercarbohydrate homologous hydrogen liaisons of the signallings of HIV and other viruses as well the functioning of the cell, producing these harmful antibodies for manufacture of medicaments /Art.52(4) EPO, Art.2(2) AT (Law 1970)/ according Claims 1,2 and 6(19,20).

Claim 10. The process of the functioning cycle of the cytoplasmic ribosomes for the folding (FKBP type of the proteins for the transporting "PKC" vesicle machinery) and the synthesis of all proteins at all ribosomes according claim 6 is characterized in that (1) During already the signal (in G1 or "G0" or the stocking signal at the G1 end), there is the constitution of the preproribosomes (nucleus , nucleole) from the proteins (with GR peptides), synthesized and folded on the active ribosomes (cytoplasm) (nuclear proteins as nucleolin or fibrillarin or ribosomal proteins as L5 or of the machinery of the transporting "PKC" vesicles /to be stocked/ or the steroid receptors that attach to the rRNA in nucleus /nucleole/); and these preproribosomes are activated (1st step!) by the special signal with the nuclear (serine or cysteine) proteinases (where the proteolysis of the N-part of the nucleolin, attached to the chromatin, is necessary for the pre-rRNA transcription) and they go to the cytoplasm where the 3'

mRNA part serves as the guide for the cytoskeletal localisation; (2) These proribosomes (cytoplasm) are already activated definitively with the cathepsin L (CL) help that cuts (at GR peptides) the particular ribosomal proteins as L5 and the nuclear proteins as the nucleolin and fibrillarin (serving as fusible) and (CL) liberates also the stocked proteins of the "PKC" transporting vesicle machinery (necessary to start again the machinery of the proteins of the "PKC" transporting vesicle cycles without their synthesis) wherein these active ribosomes make the new synthesis and foldings with the help of the corresponding chaperons (FKBP type), attached yet from the nucleus, where, naturally, all ribosomes for all proteins, have the same cycles but without "traveling" proteins with the GR peptides where the same effective process of the protein synthesis of all proteins at the proribosomes (in reality, obtained from the cytoplasm) in vitro (attached for instance on the artificial surfaces) must be made with such CL activation: proribosomes→ribosomes.

Claim 11. The manufactured medicaments /Art.52(4) EPO, Art.2(2) AT law; Accord WIPO–AT/ against the state of the clinical death and coma as phosphatidylinositol–4,5–biphosphate or its derivatives including the lysoderivatives (with easier integration in PM with transport to the interior PM layer) and, like GTP–γS (less hydrolysable substratum also for vesicle transport) characterized in that all these substances help to avoid the process of the irreversible apoptosis of the cells of the brain and heart (original reason of the state of the clinical death and coma) according Claims 1 and 6.

Claim 12. The manufactured medicaments (hypnotics) /Art.52(4) EPO, Art.2(2) AT and Accord WIPO–AT) for the partial inhibition of the cycle of the "PKC" and synaptic ("PKC"–like) vesicles (like very deluted cyanate) for the sleep process are characterized in that they cut partially the cyclic system of the neurons of the superior brain (determining the cycle, establishing the conscience), functioning by the chaotic permanent cycles of the synaptic ("PKC"– like) transport vesicles according Claim 6(12).